Hydrogenated Oil and Trans Fat's Indirect Causal Pathway Toward Elevating LDL Cholesterol

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Introduction

The link between the consumption of both hydrogenated oils and trans fats and increased serum levels of LDL and triglycerides has been well-documented, but the exact causal mechanism through which the consumption of these oils increases the amount of LDL produced by the liver remains unclear.

Contrary to common belief, these unhealthy lipids are made in the liver in an indirect response to the consumption of hydrogenated oils. These oils do not actually deliver the LDL directly into the blood stream through the digestive tract.

Abstract

There is, however, a potentially mistaken belief amongst even researchers that the hydrogenated oils in foods are entering the blood stream through the digestive tract and that the direct interaction between the oil molecules and the actual liver is somehow stimulating the LDL production. On this point, I propose that a false assumption has been made by the scientific community.

Take the case of capsaicin, which has been shown to cause almost immediate sweating around the scalp when spicy foods are consumed. This is an example of a chemical that alters the production of chemicals in the brain (specifically in the hypothalamus) *via absorption through the roof of the mouth and not through the digestive tract*. The fact that this effect has been observed and proven to exist should suggest to us that other chemicals can cross the blood-brain barrier through oral-pharyngeal osmosis and affect the functions of the body through this indirect mechanism.

I propose that hydrogenated oils are osmoted via an oral-pharyngeal route and cross the blood-brain barrier. Once within the brain, these oils trigger the production of an unidentified hormone that is released into the blood stream thereby stimulating the liver to produce LDL.

If this hypothesis can be substantiated, it would mean that we may, as a direct result of this discovery, identify the primary hormone and therefore the primary receptor in the liver critical to the process of triggering unwanted LDL production. If the brain can produce one chemical that stimulates LDL production in the liver, it stands to reason that it may also be capable of producing another chemical that shuts off that production in a natural and side-effectless manner. At minimum, the triggering hormone, if identified, could be blocked either from being produced in the brain in the first place, or from attaching to receptors in the liver.

This hypothesis may be initially validated by carrying out a study in which one group of study participants masticate and then spit out foods rich in hydrogenated oils, another group does the same but immediately and thoroughly brushes their teeth and rinses with mouthwash afterward, and in which a third group does not put hydrogenated oil into their mouths in the first place. Study participants would then undergo testing of their LDL levels a short time later to see if there is an observable change to serum LDL as a result hydrogenated oils being osmoted through the roof of the mouth.

Conclusion

If that study reinforces this notion, more intensive study of this pathway would be justifiable and a new remedy for hyperlipidemia may be ultimately identified.